PART 6

THE LUNGS

CHAPTER 28

Inspection of the Chest

KEY TEACHING POINTS

- Clubbing is best defined as either an interphalangeal depth ratio greater than 1 or positive Schamroth sign. An older definition, the hyponychial angle greater than 190 degrees, is accurate but difficult to measure at the bedside.
- The cause of clubbing is usually evident on a chest radiograph.
- In patients with cystic fibrosis, clubbing increases probability of hypoxemia; in febrile patients, clubbing increases probability of endocarditis; and in patients with cirrhosis, clubbing increases the probability of hepatopulmonary syndrome.
- Pursed lip breathing increases the probability of chronic obstructive lung disease.
- Accessory muscle use is defined as contraction of any muscle other than the diaphragm during inspiration or use of any muscle during expiration. Accessory muscle use may appear in a wide variety of respiratory disorders. When the patient is supine, the *absence* of accessory muscle use *decreases* the probability of respiratory muscle weakness.

This chapter discusses the findings of clubbing, barrel chest, pursed lip breathing, accessory muscle use, and inspiratory white noise. Other relevant findings from inspection of the respiratory system include cyanosis (Chapter 9), abnormal respiratory rate, and abnormal breathing patterns (Chapter 19).

I. CLUBBING (ACROPACHY, HIPPOCRATIC FINGERS)

A. INTRODUCTION

Clubbing is a painless focal enlargement of the connective tissue in the terminal phalanges of the digits.[1,2] Clubbing is usually symmetric, affecting fingers more prominently than toes. Although some persons have hereditary clubbing, the finding usually indicates serious underlying disease (see the section on Clinical Significance).

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Hippocrates first described clubbing in the 3rd century BC. He noted it in patients with empyema, commenting that "the fingernails become curved and the fingers become warm, especially at their tips."[3]

B. THE FINDING

Precise definitions of clubbing were developed in the 1960s and 1970s, prompted by reports that clinicians of that time were using at least a dozen different definitions[4] and by the observation that clubbing regresses after effective treatment of the underlying disorder, thus making accurate measures of this physical finding an important endpoint to follow. There are three substantiated definitions of clubbing (Fig. 28.1): (1) interphalangeal depth ratio greater than 1, (2) hyponychial angle greater than 190 degrees, and (3) positive Schamroth sign.

1. INTERPHALANGEAL DEPTH RATIO

Measurement of the interphalangeal depth ratio is described in Fig. 28.1. If this ratio exceeds 1, clubbing is present, a conclusion supported by two observations: (1) the interphalangeal depth ratio of normal persons is 0.895 ± 0.041, making the threshold

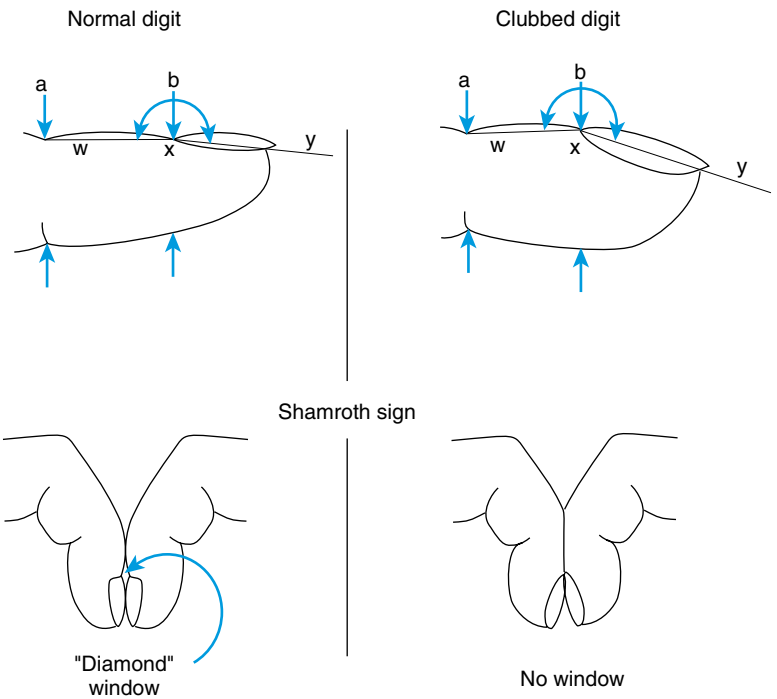

FIG. 28.1 CLUBBING. The normal digit is on the *left*, the clubbed one, on the *right*. The distal interphalangeal joint is denoted by *a;* the junction of the nail and skin at the midline is denoted by *b*. The interphalangeal depth ratio is the ratio of the digit's depth measured at *b* divided by that at *a*. The hyponychial angle is the angle *wxy*. In the figure the depth ratio is 0.9 for the normal digit and 1.2 for the clubbed digit (a ratio > 1 indicates clubbing), and the hyponychial angle is 185 degrees for the normal digit and 200 degrees for the clubbed digit (a hyponychial angle > 190 degrees indicates clubbing). The Shamroth sign refers to the absence of the diamond-shaped window that normally appears when the terminal phalanges of similar digits are opposed to each other.

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of 1 more than 2.5 standard deviations (SDs) above normal,[5,6] and (2) a ratio of 1 distinguishes digits of healthy persons from those of patients with disorders traditionally associated with clubbing (such as cyanotic heart disease and cystic fibrosis). For example, studies demonstrate that 75% to 91% of patients with cystic fibrosis have an interphalangeal depth ratio exceeding 1 but only 0% to 1.5% of normal persons do.[5,6]

2. HYPONYCHIAL ANGLE

Measurement of the hyponychial angle is described in Fig. 28.1. If this angle exceeds 190 degrees, clubbing is present, a conclusion supported by three observations: (1) the normal hyponychial angle is 180 ± 4.2 degrees, and thus the 190 degree threshold is almost 2.5 SDs above normal,[5,7,8] (2) the hyponychial angle is the best parameter distinguishing plaster casts of digits labeled "definitely clubbed" by experienced clinicians from those labeled "definitely normal,"[9] and (3) studies show that 69% to 80% of patients with cystic fibrosis have hyponychial angles exceeding 190 degrees, whereas only 0% to 1.6% of normal persons have angles this large.[7,8]

A disadvantage of the hyponychial angle is the special equipment required for precise measurements. Historically, clinicians used an apparatus called the shadowgraph, an instrument projecting the silhouette of the finger against a screen fitted with a movable protractor.[10] Modern investigators use computerized analysis of digital photographs.[8]

3. SCHAMROTH SIGN

In 1976, after watching his own clubbing come and go during an episode of endocarditis, the renowned electrocardiographer Leo Schamroth[11] suggested that clinicians place the terminal phalanges of similar fingers back to back (especially ring fingers) and look for a small diamond-shaped window outlined by the bases of nail beds and nails. Clubbing is *absent* when this window appears; clubbing is *present* when this window is missing (see Fig. 28.1). Schamroth suggested further study of his sign, and in 2010 investigators using the interphalangeal depth ratio as the diagnostic standard demonstrated that Schamroth sign had a sensitivity of 77% to 87%, specificity of 90%, positive likelihood ratio (LR) of 8, and negative LR of 0.2.[12]

4. OTHER DEFINITIONS

Parameters found to be less accurate definitions of clubbing (compared with the hyponychial angle and interphalangeal depth ratio) are the distal interphalangeal *width* ratio, the longitudinal curvature of the nail, the transverse curvature of the nail, and the profile angle (i.e., the angle between line *wx* in Fig. 28.1 and a second line extending from *x* to a point on the top of the nail approximately a third of the distance from nail fold to nail tip).[9,13]

C. CLINICAL SIGNIFICANCE

1. ETIOLOGY

In a study of 350 patients with clubbing, 80% had underlying respiratory disorders (e.g., lung tumor, bronchiectasis, lung abscess, empyema, interstitial fibrosis), 10% to 15% had miscellaneous disorders (congenital cyanotic heart disease, liver cirrhosis, chronic diarrhea, subacute endocarditis), and 5% to 10% had hereditary or idiopathic clubbing.[14]

2. RELATIONSHIP OF CLUBBING TO HYPERTROPHIC OSTEOARTHROPATHY

Clubbing may be associated with hypertrophic osteoarthropathy, a painful condition causing swelling and arthritis of the distal arms and legs. Radiographs reveal periosteal elevation of the diaphysis of long bones.[15] The usual cause is intrathoracic neoplasm (e.g., lung cancer or mesothelioma).

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3. CLUBBING AND CYSTIC FIBROSIS

In patients with cystic fibrosis, clubbing (i.e., interphalangeal depth ratio >1) predicts significant hypoxemia (i.e., PaO_2 ≤88 mm Hg on room air) with a positive LR of 3.2 and negative LR of 0.1 (EBM Box 28.1). After lung transplantation the clubbing of cystic fibrosis slowly regresses over months.[23]

4. CLUBBING AND ENDOCARDITIS

In a study of almost 2000 patients undergoing evaluation for endocarditis,[16] the finding of clubbing increased the probability of definite endocarditis (LR = 5.1; see EBM Box 28.1).

5. CLUBBING AND HEPATOPULMONARY SYNDROME

In patients with liver cirrhosis the finding of clubbing increases the probability of hepatopulmonary syndrome (LR = 4; see EBM Box 28.1; see Chapter 8).

EBM BOX 28.1
*Clubbing**

Finding (Reference)†	*Sensitivity (%)*	*Specificity (%)*	Likelihood Ratio‡ if Finding Is	
			Present	*Absent*
Detecting hypoxemia (pO_2 ≤88 mm Hg) in patients with cystic fibrosis[6]	91	72	**3.2**	**0.1**
Detecting "definite" endocarditis[16]	6	99	**5.1**	NS
Detecting hepatopulmonary syndrome in patients with cirrhosis[17-22]	22-80	64-95	**4.0**	0.5

*Diagnostic standard: for *definite endocarditis*, modified Duke criteria; for *hepatopulmonary syndrome*, triad of cirrhosis, intrapulmonary shunting by contrast echocardiography, and arterial pO_2 ≤70 mm Hg,[19] ≤80 mm Hg,[17,21] or alveolar to arterial oxygen gradient ≥15 mm Hg,[20,22] or >20 mm Hg.[18]
†Definition of findings: for *clubbing*, interphalangeal depth ratio >1,[6] or undefined.[16,17,19-22]
‡Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.
NS, Not significant.
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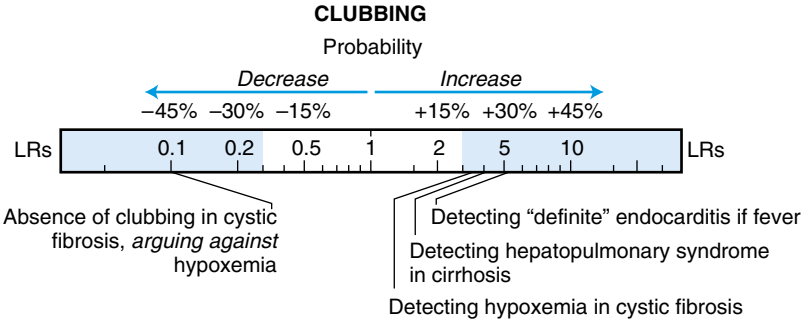

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D. PATHOGENESIS

The increased volume of the clubbed digit is primarily due to increased amounts of vascular connective tissue,[24] although the cause of this fibrovascular proliferation is still debated. According to one hypothesis, clubbing results from large megakaryocytes and clumps of platelets that become trapped in the distal digits and then release growth factors, causing soft tissue growth.[25,26] Megakaryocytes do not normally appear in arterial blood; they leave the bone marrow and travel in the systemic veins to the pulmonary capillaries, where they become trapped because of their large size (20 to 50 μm in diameter) and fragment into smaller platelets. In most patients with clubbing, the pulmonary capillaries are either damaged (e.g., as in many inflammatory and neoplastic pulmonary disorders) or a right-to-left shunt exists (e.g., as in congenital heart disease or the hepatopulmonary syndrome of cirrhosis), which allows the large megakaryocytes to travel freely through the lung into arterial blood and the distal digits, where they become wedged in the digital capillaries and release growth factors, causing fibrovascular proliferation and clubbing.

This hypothesis explains why clubbing accompanies endocarditis and why it is sometimes found unilaterally in the digits distal to an infected dialysis shunt. In both examples, platelet clumps are presumably released from the infected surface to travel to the digits, where they become embedded within capillaries and release growth factors.[25]

An alternative hypothesis (though not necessarily a contradictory one) proposes that clubbing stems from elevated levels of prostaglandin E2 (PGE_2). In families of patients with hereditary clubbing and osteoarthropathy, defective catabolism of PGE_2 causes high levels of this PGE_2 to accumulate.[27]

II. BARREL CHEST

A. THE FINDING

The normal chest is shaped like an oval cylinder, its anteroposterior diameter being less than its lateral diameter. The ratio of the anteroposterior to lateral diameter (called the *thoracic ratio*, *thoracic index*, or *chest index*) is normally approximately 0.70 to 0.75 in adults and increases as persons grow older. The upper normal limit is approximately 0.9.[28]

Barrel chest deformity refers to a chest whose transverse section is more round than oval. It is traditionally a finding of chronic obstructive lung disease (i.e., chronic bronchitis, emphysema). Most patients also have dorsal kyphosis, a prominent sternum, widened intercostal spaces, elevated clavicles, and a shortened neck.[28] According to traditional teachings the thoracic ratio of these patients exceeds 0.9, presumably because overactive scalene and sternocleidomastoid muscles lift the upper ribs and sternum (see the section on Accessory Muscle Use).

B. CLINICAL SIGNIFICANCE

Evidence linking the barrel chest deformity with chronic obstructive lung disease is conflicting. Two studies did find a significant correlation between the barrel chest deformity and more severe airflow obstruction,[29,30] although another two studies found no relationship between the two conditions.[28,31] Additional problems with this physical sign are that the barrel chest is not specific for obstruction but also occurs in elderly persons without lung disease.[28] In some patients the large anteroposterior dimension of the barrel chest is an illusion; the actual

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anteroposterior dimension is normal but it appears to be abnormally large because it contrasts with an abnormally thin abdominal dimension caused by weight loss (Fig. 28.2).[32]

In a single study the presence of a barrel chest, defined either as clinician's global impression of barrel chest or more precisely as a thoracic ratio greater than or equal to 0.9, modestly increased the probability of obstructive disease (LRs = 1.5 to 2.0, EBM Box 28.2).

III. PURSED LIP BREATHING

A. THE FINDING

Many patients with chronic obstructive lung disease instinctively learn that pursing the lips during expiration reduces dyspnea. The exact cause of the relief of dyspnea is still debated. Pursed lip breathing significantly reduces the respiratory rate (from approximately 20 breaths/minute to 12 to 15 breaths/minute), increases tidal volume (by approximately 250 to 800 mL), decreases $PaCO_2$ (by 5%), and increases oxygen saturation (by 3%).[36-39] Dyspnea may diminish because there is less work of breathing (from slower rate), less expiratory airway collapse (the pressure drop across the lips, 2 to 4 cm of water, provides continuous expiratory positive pressure), or recruitment of respiratory muscles in a way that is less fatiguing to the diaphragm.[36,37,40]

B. CLINICAL SIGNIFICANCE

In a study of 200 patients presenting for pulmonary function tests, the finding of pursed lip breathing increased the probability of chronic obstructive disease (LR = 2.7).

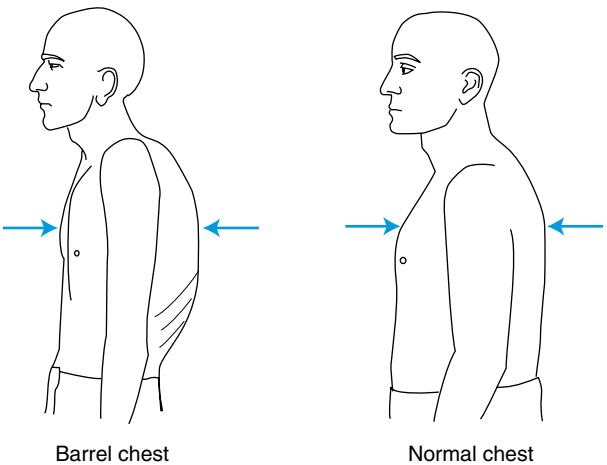

FIG. 28.2 BARREL CHEST DEFORMITY. In some patients the "large" anteroposterior dimension of the barrel chest (*left*) is an illusion because it is no bigger that the anteroposterior dimension of the normal chest (*right*). Instead, what strikes the clinician's eyes is the barrel chest's prominent dorsal kyphosis and marked contrast between the preserved anteroposterior chest dimension and the thin abdomen.

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EBM BOX 28.2
*Inspection of the Chest**

Finding (Reference)†	*Sensitivity (%)*	*Specificity (%)*	Likelihood Ratio‡ if Finding Is *Present*	Likelihood Ratio‡ if Finding Is *Absent*
Chest Wall Appearance				
Barrel chest, detecting chronic obstructive lung disease[33]	65	58	1.5	0.6
AP/L chest diameter ratio ≥0.9, detecting chronic obstructive lung disease[33]	31	84	2.0	NS
Pursed Lip Breathing				
Pursed lip breathing, detecting chronic obstructive lung disease[33]	58	78	2.7	0.5
Accessory Muscle Use				
Scalene/sternocleidomastoid muscle use, detecting chronic obstructive lung disease[33]	39	88	**3.3**	0.7
Scalene/sternocleidomastoid muscle use in patients with amyotrophic lateral sclerosis, detecting respiratory neuromuscular weakness[34]	81	83	NS	**0.2**
Accessory muscle use, detecting pulmonary embolism[35]	17	89	NS	NS

*Diagnostic standard: for *chronic obstructive lung disease*, FEV1/FVC <0.7; for *respiratory neuromuscular weakness*, transdiaphragmatic sniff pressure <70 cm H_2O; and for *pulmonary embolism*, pulmonary angiogram.
†Definition of findings: for *accessory muscle use* in patients with amyotrophic lateral sclerosis, the patients were examined supine.
‡Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.
AP/L, Ratio of anteroposterior chest dimension to lateral dimension; *FEV1*, forced expiratory volume in 1 second; *FVC*, forced vital capacity; *NS*, not significant.
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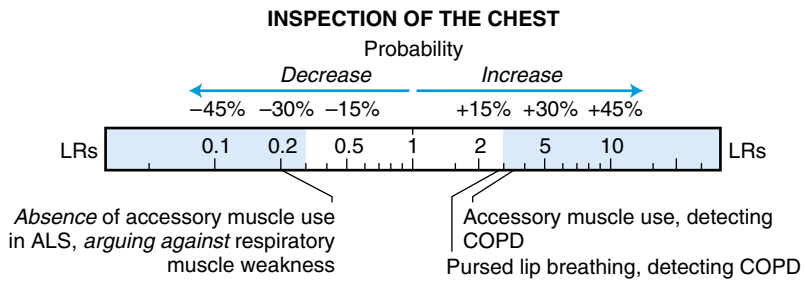

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IV. ACCESSORY MUSCLE USE

A. THE FINDING

The only muscle used in normal breathing is the diaphragm, which contracts during inspiration. Normal expiration is a passive process that relies on the elastic recoil of the lungs.[41] Therefore the term **accessory muscle use** refers to the contraction of muscles other than the diaphragm during inspiration (usually the sternocleidomastoid and scalene muscles) or to the contraction of any muscle during expiration (primarily the abdominal oblique muscles). Accessory muscle use is a common finding in patients with chronic obstructive lung disease or respiratory muscle fatigue.

B. PATHOGENESIS

Contraction of the sternocleidomastoid and scalene muscles lifts the clavicles and first ribs, which helps to expand the thorax of distressed patients, especially those with chronic obstructive lung disease whose flattened diaphragm generates only meager inspiratory movements. Contraction of the abdominal oblique muscles assists ventilation in two ways. In patients with obstructed airways, the abdominal muscles help to expel air across the obstructed airways; in patients with respiratory muscle fatigue (e.g., amyotrophic lateral sclerosis), the abdominal muscles characteristically contract right at the moment expiration ends, to compress the lungs so that the early part of the subsequent inspiration can occur passively.[42]

C. CLINICAL SIGNIFICANCE

Accessory muscle use—defined as inspiratory contraction of the sternocleidomastoid and scalene muscles—is associated with severe obstructive disease.[29,31,43-45] More than 90% of patients hospitalized with acute exacerbations of chronic obstructive lung disease use accessory muscles, but by hospital day 5, less than half do.[46] In one study, patients whose clavicle lifted more than 5 mm during inspiration identified patients with more severe obstructive disease (mean forced expiratory volume in one second [FEV1] = 0.6 L vs. 1.5 L; $p < 0.001$),*[43] and in patients referred for pulmonary function tests, accessory muscle use increases the probability of chronic obstructive disease (LR = 3.3; see EBM Box 28.2).

Inspection of accessory muscles also provides useful information in patients with amyotrophic lateral sclerosis. When these patients are supine, the *absence* of sternocleidomastoid and scalene contractions decreases the probability of respiratory neuromuscular weakness (LR = 0.2).

Accessory muscle use is less specific in the evaluation of acute dyspnea, and in one study of patients with suspected pulmonary embolism, the finding had no diagnostic value (see EBM Box 28.2).

*FEV1 is forced expiratory volume in one second, a measure of ventilatory capacity. Normal values are 3 to 3.8 L.[47] The FEV1 is abnormally low in obstructive lung disease and restrictive lung disease, dyspnea first appearing in these conditions when the FEV1 falls below 2.5 L. An FEV1 less than 1 L in chronic obstructive lung disease indicates severe disease.

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V. INTENSITY OF BREATHING SOUNDS (INSPIRATORY WHITE NOISE; NOISY BREATHING)

A. THE FINDING

The breathing of normal persons is inaudible more than a few centimeters from the mouth, unless the person is sighing, panting, or gasping.[48] In three clinical settings, breathing sometimes becomes very noisy and is easily heard a distance from the bedside: in patients with lower airways obstruction, who may have audible *expiratory* wheezing (see Chapter 30), in patients with upper airway obstruction, who may have *inspiratory* stridor (see Chapter 30), and in patients with chronic bronchitis or asthma, who may have *inspiratory* white noise.[48]

White noise is an acoustical term. Unlike wheezing and stridor, white noise lacks a musical pitch and therefore resembles more the static of a radio tuned between stations. In patients with chronic bronchitis and asthma the loud inspiratory white noise heard at the patient's bedside without the stethoscope often contrasts sharply with the quiet inspiratory sounds heard through the stethoscope during auscultation (see Chapter 30).

B. PATHOGENESIS

Inspiratory white noise results from air turbulence caused by narrowed central airways,[49] a conclusion based on the observation that the sounds diminish after the patient receives effective bronchodilator treatment (which increases the patient's FEV1) or breathes a mixture of oxygen and helium (a gas mixture that reduces turbulence).[49] Inspiratory white noise is not a feature of emphysema, presumably because the inspiratory caliber of the central airways in these patients is normal.[49]

C. CLINICAL SIGNIFICANCE

Inspiratory white noise is a feature of chronic bronchitis and asthma, not emphysema. The intensity of white noise in patients with asthma and chronic bronchitis correlates inversely with the patient's FEV1 ($r = -0.60$ to -0.64).[49]

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